# A Study on the Preventive and Therapeutic Effects of Curcumin on Non-Alcoholic Fatty Liver Disease

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**Abstract.** Non-alcoholic fatty liver disease (NAFLD) has become increasingly common worldwide and now accounts for a substantial burden of chronic liver disease. In some patients, hepatic steatosis progresses to nonalcoholic steatohepatitis, cirrhosis, and eventually liver cancer. Because treatment choices remain limited and some available therapies raise concerns about adverse effects or tolerability, compounds with a favorable safety profile continue to receive attention. Curcumin, the principal bioactive constituent of turmeric, has been investigated in this context. The evidence summarized in this review links curcumin with less hepatic fat deposition and fibrotic change, together with improvement in abnormal glucose and lipid metabolism. The discussion is organized around three closely related aspects of NAFLD: disturbance of lipid metabolism, hepatic inflammation, and fibrosis. It further considers why these experimental and clinical observations have not yet translated readily into treatment, with particular attention to poor absorption and low bioavailability. Possible directions for subsequent work, including combination approaches, are also discussed.

**Keywords**: curcumin, non-alcoholic fatty liver disease, nonalcoholic steatohepatitis

## 1 Introduction

After excessive alcohol consumption (≥30 g/day for men and ≥20 g/day for women) and other secondary causes of liver disease have been excluded, hepatic steatosis above 5%, assessed by liver biopsy or magnetic resonance imaging proton density fat fraction (MRI PDFF), is used here as the diagnostic basis for NAFLD [1, 2]. The condition has become increasingly common and now represents an important public health problem. The estimate cited in this review places the prevalence in adults at nearly 25%, while rates among adolescents have also been reported to rise over time [3]. Disease severity is variable: NAFLD may remain as steatosis, but it can also advance to nonalcoholic steatohepatitis (NASH), cirrhosis, or liver cancer; a figure of 15% to 20% has been reported for disease progression in the cited literature. In China, NAFLD contributes substantially to the burden of chronic liver disease, whereas in the United States it is also prominent among conditions associated with liver transplantation [4, 5]. Its de-

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velopment involves multiple interacting mechanisms rather than a single pathological process. The literature considered in this paper also describes limitations in current treatment, including the absence of a specific drug for NAFLD in the cited context and tolerability or adverse-effect concerns with agents such as insulin sensitizers and statins. These problems make early intervention important and provide a rationale for continuing to investigate therapies that combine efficacy with acceptable safety [6, 7].

Curcumin is the major bioactive constituent of turmeric and has been studied for a broad range of pharmacological activities. Reported effects include anti-inflammatory, antioxidant, antiviral, glucose-lowering, lipid-regulating, anticancer, and free-radical-scavenging actions [8]. Within liver disease research, particular attention has been given to its effects on hepatic steatosis and fibrotic change. The metabolic relevance of curcumin is not limited to the liver; its influence on disturbed glucose and lipid homeostasis has also been examined in insulin resistance, obesity, and diabetes [8, 9]. Taken together, these observations provide the basis for considering curcumin as a possible means of alleviating manifestations of NAFLD. The exact molecular events responsible for these effects, however, remain incompletely defined, and the extent to which the reported benefits can be translated into therapeutic use still requires further study [10, 11].

## 2 Mechanism of Action of Curcumin in Improving NAFLD

### 2.1 Regulate Lipid Metabolism (Fatty Liver)

Steatosis is one of the defining pathological features of NAFLD and is associated with hepatic lipid accumulation of 5% or more [2, 12]. Curcumin may influence this process at several points in lipid handling. One route is a reduction in the amount of free fatty acid (FFA) entering hepatocytes: lower expression of fatty acid transporters decreases the FFA pool available for esterification and subsequent triglyceride formation. A second route involves citrate metabolism. When hepatic citrate availability is reduced, less acetyl-CoA is generated for triglyceride synthesis [5, 6]. Acting together, these changes can lessen triglyceride deposition and the inflammation associated with lipid overload, while also restraining hepatic stellate cell activation and helping to preserve extracellular matrix homeostasis [13]. Evidence from metabolic studies is consistent with this pattern. The analysis reported by Wei et al. found decreases in plasma low-density lipoprotein cholesterol (LDL-C), triglycerides, fasting blood glucose, and body weight in curcumin-treated groups relative to placebo [14]. Jang et al. likewise reported lower circulating and hepatic lipid levels after curcumin treatment in a high-fat-fed animal model [15].

**Inhibit de novo lipid synthesis.** Another part of the lipid-lowering effect concerns the formation of new fatty acids and triglycerides within the liver. Curcumin has been reported to alter both gene and protein expression of ATP-citrate lyase (ACLY), acetyl-CoA carboxylase (ACC), and fatty acid synthase (FAS), three enzymes that occupy important positions in this pathway. For the corresponding genes, reductions of 95%,

50%, and 77%, respectively, were reported [16]. Effects on ACLY-related metabolism may further decrease the hepatic supply of citrate used as an indirect carbon source for lipid synthesis. The same section of the literature also describes antioxidant effects: in Wistar rats, increased superoxide dismutase 1 (SOD1) expression was accompanied by stronger hepatocellular antioxidant capacity and slower development of NASH. A separate regulatory route involves AMP-activated protein kinase (AMPK). In high-fat-fed animal models, activation of hepatic AMPK by curcumin has been associated with changes in sterol regulatory element-binding proteins (SREBPs) and ACC phosphorylation. The downstream consequence is reduced synthesis of fatty acids and cholesterol, which in turn limits lipid accumulation in the liver [17, 18].

Cytosolic citrate can be supplied through the mitochondrial transporter SLC25A1, the plasma-membrane transporter SLC13A5, and the breakdown of glutamine. In the studies cited here, curcumin was associated with reduced hepatic lipid deposition through suppression of SLC13A5-dependent citrate transport together with changes in the abundance of the transporter protein [19].

**Reduce hepatic fatty acid uptake.** The movement of FFAs into hepatocytes depends on several transport-related proteins, among them fatty acid translocase (FAT/CD36), fatty acid transporters (FATPs, especially FATP2/5), and fatty acid-binding protein 1 (FABP1). In NAFLD mice, treatment with aqueous turmeric extracts was reported to decrease hepatic expression of these proteins. Work on polyethylene glycol derivatives of curcumin points to a related mechanism: activation of cyclic AMP (cAMP) was accompanied by lower CD36 expression and improvement of steatosis [20]. Cell and animal experiments have also described a reduction in both gene and protein expression of hepatic fatty acid transporters after curcumin exposure, with a corresponding decrease in FFA uptake. Other regulatory changes involve peroxisome proliferator-activated receptors (PPARs) and liver X receptor α (LXRα), whose expression can be increased by curcumin. At the same time, suppression of SREBP1 lowers the activity of lipogenic enzymes such as ACC and FAS. Enhancement of fatty acid oxidation adds another mechanism by which lipid accumulation may be reduced [21].

**Reduce lipid transfer to the liver.** Curcumin can also act before FFAs reach the liver by modifying lipid mobilization in adipose tissue. Suppression of the protein kinase A (PKA)/hormone-sensitive lipase (HSL) pathway, together with relief of endoplasmic reticulum stress, reduces adipose lipolysis. As a result, less glycerol and fewer FFAs are released into the circulation and delivered to the liver [22]. A further effect has been described through the AMPK-mediated mTOR pathway. By reducing adipose hypoxia and HIF-1α-associated fibrotic change, curcumin can improve adipose tissue function, including the control of lipolysis. This provides an additional explanation for the lower ectopic transfer of FFAs from adipose tissue to the liver [23].

**Promote fatty acid utilization.** Curcumin may additionally reduce lipid burden by favoring fatty acid use rather than storage. Increased activity or expression of hepatic CPT has been reported after curcumin treatment and is associated with lower lipid accumulation [24]. In adipose tissue, higher CPT expression promotes lipid oxidation while decreasing fatty acid esterification. Upregulation of PPARα provides a related

route for increasing fatty acid oxidation and, consequently, reducing fat deposition [25].

### 2.2 Alleviate Liver Inflammation

The transition from NAFLD to NASH is closely associated with sustained inflammatory signaling in the liver. The stimuli that maintain this response may originate within the liver or from extrahepatic sources. Among the cells involved, activated Kupffer cells and hepatic stellate cells contribute to the inflammatory environment through the production or increased expression of TNF-α, interleukin-6 (IL-6), and IL-1β [20, 26]. Curcumin is reported to act at more than one point in this network rather than on a single inflammatory target. Enzymes such as cyclooxygenase-2 (COX-2), lipoxygenase (LOX), and inducible nitric oxide synthase (iNOS) can be inhibited, while signaling mediated by nuclear factor kappa-B (NF-κB) and signal transducer and activator of transcription 3 (STAT3) is also suppressed [25]. Another reported pathway involves HIF-1α: inhibition of this factor has been associated with lower hepatic IL-6 and TNF-α expression and higher expression of the anti-inflammatory cytokine IL-10 following curcumin treatment [27].

At the hepatocyte level, the reported anti-inflammatory action of curcumin involves both inflammatory signaling and oxidative stress. Curcumin reduces the activity as well as the gene and protein expression of several signaling components, thereby decreasing the release of chemokines and other inflammatory mediators and limiting inflammatory-cell activation and proliferation. In parallel, excess hepatic reactive oxygen species (ROS) are removed more effectively, SOD activity rises, and malondialdehyde (MDA) levels fall. The combined effect is a reduction in oxidative stress and a weaker inflammatory response [28]. SIRT1-dependent regulation provides another link between these processes: increased SOD1 expression can relieve endoplasmic reticulum stress and thereby attenuate steato-inflammatory changes in the liver [29].

### 2.3 Anti-Hepatic Fibrosis

Fibrotic progression in fatty liver disease is favored by persistent inflammatory injury and continued activation of hepatic stellate cells. Within this setting, curcumin has been reported to reduce insulin signaling and oxidative stress, with a consequent decrease in genes associated with stellate-cell activation, including type I collagen (Col1). Lower expression of these activation-related genes is accompanied by less extracellular matrix deposition [30]. Curcumin can also restrain stellate-cell activation and proliferation through inhibition of leptin signaling, enhancement of PPARγ activity, and further reduction of oxidative stress [31]. Related antifibrotic effects have been described outside the liver as well. In adipose tissue, modulation of the AMPK-mediated mTOR pathway decreases expression of genes encoding type I collagen (Col1α), type III collagen (Col3α), and type VI collagen (Col6α), which helps alleviate fibrosis induced by a high-fat diet [23].

## 3 Conclusions and Prospects

Taken together, the studies reviewed here point to three recurring effects of curcumin in NAFLD: a reduction in hepatic lipid deposition, better control of oxidative-stress products, and less accumulation of extracellular matrix. By acting on these processes, curcumin may interfere with several changes that accompany the development and progression of non-alcoholic fatty liver disease.

The available findings therefore support continued evaluation of curcumin in NAFLD and other metabolic disorders, and they also provide a pharmacological basis for exploring the modernization of traditional Chinese medicine. Important uncertainties remain. The molecular actions of curcumin have not been fully clarified, and its clinical effectiveness and safety require more evidence. Future studies of genes, epigenetic regulation, and adipokines may help explain parts of NAFLD pathogenesis that remain unresolved [32]. Mitochondria-directed strategies represent another area of interest, although problems related to immunogenicity and stability still complicate their translation into clinical use [7, 33]. Combination treatment may be useful when a single intervention is insufficient, while targeted delivery could improve exposure at relevant tissues and reduce unnecessary systemic effects. The multi-target and multi-pathway actions attributed to curcumin provide a reason for continued investigation [34], but poor bioavailability remains an important limitation. Subsequent work should therefore address formulation and delivery as well as longer-term treatment performance, while also establishing safe and standardized dosing approaches [10, 35].

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